A CASE FOR DIAGNOSIS DECOMPRESSION SICKNESS OR MIGRAINE?

Chris Lourey

A 39 year old male, an ex-professional diver with 20 years experience, so-called, presented at Casualty after a dive complaining of blurring of vision, pain and skin rash in the epigastrium, lethargy and dizziness. He had focal but resolving right sided neurological symptoms and signs. These were pain, paraesthesiae and weakness of right arm.

He confidently stated that he was in Casualty only because of his wife's insistence, and that he had suffered two episodes of skin rashes after diving in the past and that this was all due to a migraine. He has 4 to 6 migraine attacks a year.

At all times until discharge the patient maintained an aggressive and at times paranoid mental state. There were gross discrepancies in data and detail between his diving buddy, the patient and his wife's testimony.

His diving profile, psycho-social-physical status and clinical management make for interesting discussion.

THE DIVE

He dived approximately half a mile east of the Nobbies in Bass Strait, using compressed air from a compressor to dual hookah hoses. He wore a 5/16th inch wet suit.

The visibility was approximately 8 to 10 ft. The water temperature was such that both divers felt colder than usual. There was a 15 to 20 foot swell. The depth was said to be 10 to 50 ft. However the relevant charts show bottom depths up to 80 ft or more in this area.

On the evening before the dive and just before the dive he had taken two "Orthoxicol" capsules for a head cold.

He entered the water at 1100 and did bottom sweeps till 1145 when he surfaced with a bag full of fish. From 1150 till 1230 he performed more bottom sweeps. He left the water at 1230. From 1235 till 1300 he had coffee and a few cigarettes. He re-entered the water at 1300 and performed more bottom sweeps until 1430 when he returned to the boat, without decompression stops. He then had lunch of beer, coffee and sandwiches and cigarettes.

At 1515 he noticed a headache, fatigue and blurring of vision. An hour later there had been no change in the symptoms. However he had now developed a rash which was more extensive than when he appeared at Casualty. By 1645 he felt giddy and disorientated. The blurring of vision increased. He lay down in the cabin and noticed weakness of the right arm with paraesthesiae and pain in the right elbow.

After approximately 60 minutes the paraesthesiae abated slowly, but the weakness and blurring of vision, although resolving, remained until approximately 8 hours after admission.

CLINICAL MANAGEMENT

Condition on arrival at hospital

On admission he had a generalised rash, particularly on the trunk and torso. His blood pressure was 150/90 lying, and 80/60 standing. His pulse rate was 96/min. There was no cranial nerve deficit nor any nystagmus. Tests of vision and visual fields were normal as were the fundi. Cerebellar function was normal. There was a very mild weakness of the right arm, but no specific neural deficit. The plantar reflexes were normal. Clonus could not be elicited. Otological examination was normal except for bilateral exostoses. Auscultation of the chest was normal except for occasional mild basal wheezes. He had no difficulty in voiding. No other specific abnormality noted.

His concentration was poor.

Investigations

Chest X-ray was normal except for mild hyper-inflation. There was evidence of mediastinal or subcutaneous emphysema.

Blood gases were done while breathing air. The results were pH 7.39, PO_2 77mmHg, PCO_2 36mmHg, HCO_3 22, Base Excess -2.

A full blood examination was performed. The results were Hb 17gm%, PCV 0.55, WCC 16,300, Platelets 80.3 x 10^3.

The electrolytes etc. were Na 141, K4.3, Urea 4.9, Glucose 4.8.

Fibrin degradation products were not tested as there are no facilities for this at Frankston Hospital.

Cardiac enzymes were within normal limits and remained so.

ECG showed first degree heart attack with ST elevation in Leads II and V6. The T wave in V1 was greater than in V6, ie. evidence of pericardial and/or epicardial injury pattern.

A CAT scan of the head and cranial region to C5 was normal.

An EEG was not performed.

Diagnosis

The diagnosis was obviously decompression sickness with skin, pericardial and possibly epicardial involvement and neurological manifestations.

Migraine as the aetiological factor of the waning hemiparesis and visual disturbance was also considered.

Myocardial infarction was considered and investigated.

He was treated with

1. IV-fluids were started with 3 litres of Balanced Salt Solution over 3 hours then maintenance therapy. This regime was empirically based as a CVP line was not inserted because of the low platelet count.

2. 100% oxygen was given for 6 hours then 35% was given for 48 hours with 6 hourly periods of 1 hour on 100% oxygen.

3. Dexamethazone.

4. Salicylate therapy was started with 600 mg of aspirin stat, then 300 mg daily for 4 days.

5. Routine oral antacid therapy.

6. Continuous ECG monitoring and serial enzyme assays.

The National Safety Council of Australia, Victorian Division (NSCA. (Vic)) at Morwell were phoned after institut-

ing 1 and 2 above, for possible transfer for recompression and hyperbaric oxygen (HBO) therapy. However they considered as there had been neurological improvement, migraine was the likely aetiological basis for his transient visual disturbance and hemiparesis, and that the transfer of 130 kilometres at midnight not warranted. It was recommended that if any deterioration occurred he should be transferred to Morwell for recompression.

Treatments 3, 4, 5 and 6 were then instituted. He gradually improved over the next 24 hours, but his lethargy and minor degree of mental dullness remained, and improved slowly, over the next 3 days.

His ECG returned to normal within 48 hours. One's "gut" feeling at the time was that recompression and HBO would have been worthwhile, particularly in view of the slow resolution of his mental dullness.

He failed to appear for the scheduled follow up which was to include EEG, neurological and psychological testing!

PSYCHO-SOCIAL-PHYSIOLOGICAL PROFILE

He is an ex-professional diver of 20 years experience who reluctantly admitted to previous incidents of decompression sickness. He has had migraine for more than 10 years. He is prone to extreme swings of mood. He is a heavy smoker and heavy drinker who is currently on wife number three. He has had many motor vehicle accidents (MVA) and has written off three cars. The last MVA was one week before this dive.

His diving behaviour and knowledge was at variance with safe diving practice despite his professed experience.

It is tempting to categorise this individual as a "punch drunk diver".

SUMMARY

A 39 year old male diver who presented with skin rash, epigastric pain and resolving hemiparesis.

The conservative therapeutic approach taken invites comment as does the problem of migraine and diving.

I would dispute the statement made earlier that aspirin has NO place in the treatment of DCS. However is it not a panacea. I believe that it has a role in association with the appropriate adjuvant therapy in certain clinical situations, eg. remote geographical situations.

DISCUSSION

Chairman: Dr David Brownbill

First I would like to emphasise that when central nervous tissue is damaged and dies it is not replaced. There is no repair. So when you are looking at something that may involve death of neurones you are on a downhill slide, there is no way back from that death. When observations of neurological symptoms change then that indicates that there is a pathological occurrence which may be reversible. It may not be serious but there is a pathological occurrence. One has to assess whether that occurrence is incidental and reversible such as migraine or whether it is a potentially destructive thing such as decompression sickness. In that situation one must be considering all the time the risks of missing something that is potentially dangerous as opposed to the risks of instigating the appropriate treatment, here it would have been recompression, for that condition. What is the risk of recompressing a patient with migraine as opposed to leaving someone on observation only, who may have on-going damage to cerebral structures. I think that is fundamental in the assessment of this case report and I would like to hear a lot of discussion about the approach to not what is definitely decompression sickness, but what reasonably might be.

The prime treatment for decompression sickness is recompression. The indications for treatment of central nervous system decompression sickness is not only the diagnosis of established CNS decompression sickness, but rather the diagnosis of what might reasonably be CNS decompression sickness. It is better to find that one has recompressed unnecessarily than to have sat on neuronal damage, which at first may have been reversible.

Dr Chris Lourey

I would like to ask others their thoughts on salicylate therapy. My belief is that it is used in neurological disease. In a situation such I was faced with I would still maintain that you would use it until there was properly documented data that suggests that you do otherwise.

Chairman: Dr David Brownbill

I also subscribe to the view that until something that is not dangerous is proven to be totally ineffective, then consideration should certainly be given to using it. There is no doubt that one's clinical experience, eg. in transient ischaemic attacks, that low dose aspirin is of benefit in sometimes turning off showers of transient ischaemic attacks. There is benefit in preventing the extension of problems with established embolism. Whether overall statistically aspirin is of benefit, we do not know at the moment. We will not know the answer to this until the international survey comes through. But we do know that there are specific incidences where it seems irrefutable that it has helped. On that, I would subscribe to the idea that aspirin is worthy of consideration. I know Carl Edmonds says that the person who was the protagonist of aspirin for decompression sickness is now unhappy. I do not think that the use of aspirin in neurological conditions rests with that gentleman. It has extended across the world in other neurological areas of investigation and management, so it would seem to me that there is a case at the moment of considering its use.

Dr Carl Edmonds

The man had an obvious bend. He was badly treated and you were fiddling around with aspirin and the poor man needed oxygen and a chamber. The fact that he was aggressive means that he had brain damage. It is tragic to me that he was not recompressed.

Dr John Hayman

I would like to make a plea that when we do blood tests on such patients we should also include a coagulation profile. We should be doing things like the partial thromboplastin time and fibrin degradation products both in urine and in blood. Then I think we have got some background as to what is going on and we can see the effects of aspirin and any other anticoagulant on these parameters.

Chairman: Dr David Brownbill

Here we have a story of the subtler changes of cerebral function, that is cerebration. That raises another question, whether subtle changes in mental state should not be indications for recompression. Ian Millar has a story to illustrate this point.

Dr Ian Millar

This case report concerns a patient who presented quite a diagnostic dilemma. We believe the results of her test of recompression indicate that she was suffering from cerebral decompression sickness and that this case reinforces the value of treatment upon suspicion.

A thirty-five year old experienced female sports diver had undertaken repetitive dives to 75 feet for 40 minutes followed several hours later by 30 feet for 40 minutes. That evening, along with three of her colleagues, she had suffered nausea, stomach cramps, some diarrhoea and a generalised skin rash which was thought possibly related to some seafood eaten on the dive trip. Upon her return home she felt more tired than usual, somewhat "off colour" and had a persisting skin rash. She presented to her general practitioner late the following morning with a quite obvious blotchy skin rash and significant oedema. She felt unwell and a little dizzy. She was slightly ataxic. Her GP also noticed her to be dull, quiet and not her usual self.

She had been treated with hydro-cortisone and promethazine by the time I was contacted, and her oedema and rash had settled. She had also had some oxygen over about one hour. Her cerebration was still not quite normal however, and after some deliberation she was referred down to us. When I saw her, she seemed quite a reasonable person who stated that she felt something of a fraud presenting for treatment. She was, however, rather "slow". Not having met her before, this could have been interpreted as her normal mental state, however, I had been informed that this was not so. She had very objective findings apart from poor balance with inability to maintain balance with her eyes closed. After 20 minutes at 18 metres on oxygen, her personality changed completely from being a rather dull woman with a very flat effect, to one of the brightest personalities we have had in the chamber. It became a most entertaining treatment from then, with the repartee passing in and out of the chamber. She had changed personality completely and maintained that change back to what all her friends and her doctor said was her normal self.

Dr Carl Edmonds

There is available to people who are amateurs in diving medicine a very professional service run by the Royal Australian Navy. It is called the Diver Emergency Service. It means that anyone who is an amateur medico in the field of diving medicine can phone up and speak to a really good professional and get help and advice. The number throughout Australia is 960-0321 and it is available 24 hours a day. It has been there many years but at least now it has been made widely known. I think, in the future, that will seem a sensible number to anyone, even those who are professionals, to use.

On the subject of permanent damage from CNS decompression sickness, there is a fascinating case of the man who had a very slight weakness in the left leg. He had a little pain around the back, very little, and I think that was all. He was treated very effectively in one of the Royal Navy's chambers with oxygen and recovered completely. He walked out of his house a day later and was shot dead. The neuropathology showed lesions throughout both the brain and spinal cord, even after such minimal signs.

I would like to mention something else that people may not know. About 5 years ago there was a whole series of US Navy Tables, Tables 5, 5A, 6 and 6A. Nowadays almost all treatments are with Table 6.

Chairman: Dr David Brownbill

It would seem that people who have had a central nervous tissue hit really should be considering seriously not diving again because even though there appears to be complete clinical resolution, it has now been established that pathologically this is not so.

Question

When you say there is damage done, 24 hours later, what are you looking at? Are you looking at things like congestion and oedema or are you looking at something which indicates that you should be treating these people longer? Or are you looking at something that is a permanent damage?

Dr Carl Edmonds

In that particular pathology, it was autopsy pathology and largely brain and spinal cord. As to whether you should treat people longer, nowadays, in Australia anyway, people do not have just one treatment. They have many treatments for decompression sickness. In the old days one did not do that, one sent them out after 24 hours on oxygen. Nowadays we tend to do a number of treatments. Whether this is of value or not, I do not know. Whether or not one is going to show an enormous amount of pathology eventually when someone, like that guy, coincidentally dies, we are going to have to rely on people like John Hayman.

Dr Mary Novak

Was your patient's labile blood pressure due to fluid depletion?

Dr Chris Lourey

My estimate is that it was a combination of the dehydration of cold diuresis with the diuresis of immersion with extravasation of fluid due to endothelial disruption that was responsible.

Question

Did the ECG changes revert to normal?

Dr Chris Lourey

The ECG eventually did return to normal over the next 3 days. The ST elevations gradually resumed their normal profile. The only thing that did not return to normal was that he had a first degree heart block. We were hoping to look at that at a later stage. I would also like to stress that at no stage were his enzymes elevated, which I think is a consideration in a 39 year old individual who has an exhausting dive and who comes in with upper abdominal pain or chest pain.

Dr Ian Millar

Diving Emergency calls to NSCA are now referred to one of our three full-time diving medical officers. We have developed a close relationship with the RAN School of Underwater Medicine and quite often consult them concerning difficult or unusual cases.